Changes in systemic vascular resistance detected by the arterial resistometer: preliminary report of a new method tested during percutaneous transluminal coronary angioplasty

Rephael Mohr, M.D., Shmuel Rath, M.D., Ofer Meir, Aram Smolinsky, M.D., Yedael Har-Zahav, M.D., Henry N. Neufeld, M.D., and Daniel A. Goor, M.D.

ABSTRACT A recently developed apparatus provides on-line continuous monitoring of systemic vascular resistance (SVR) by means of simple computer analysis of the peripheral arterial waveform. The fundamental equation of this method is \( Ri = \frac{P'}{(dP/dt)} \), where \( dP/dt \) is the peak \( dP/dt \) of the peripheral arterial waveform, \( P' \) is the pressure at time of peak \( dP/dt \), and \( Ri \) is a resistance index that bears a direct relation to SVR. Eleven patients undergoing percutaneous transluminal coronary angioplasty (PTCA) were studied to evaluate the changes in SVR associated with myocardial ischemia (angina detection). There were 49 balloon inflations, all of which were associated with an increase in \( Ri \) (from 38.4 \( \pm \) 12 to 81.2 \( \pm \) 36 \( \times \) 10\(^{-3} \) sec; \( p < .01 \)) and a decrease in \( dP/dt \) (from 2076 \( \pm \) 257 to 1327 \( \pm \) 326 mm Hg/sec; \( p < .01 \)). In 42 of the balloon inflations these changes were associated with electrocardiographic ST-T changes and in 23 it was also associated with anginal pain. When angina was present, a further increase in \( Ri \) (to 97.5 \( \pm \) 43 \( \times \) 10\(^{-3} \) sec; \( p < .01 \)) and a decrease in \( dP/dt \) (to 1218 \( \pm \) 338 mm Hg/sec; \( p < .01 \)) was observed. It was found that myocardial ischemia is associated with an increase in the resistance index and a decrease in \( dP/dt \) and can be detected by the resistometer.

Circulation 74, No. 4, 780-785, 1986.

WE HAVE RECENTLY SHOWN that a simple computer analysis of the peripheral arterial waveform can provide on-line continuous monitoring of systemic vascular resistance (SVR).\(^1\)\(^2\) The apparatus that monitors these changes is called the arterial resistometer.\(^2\)

This apparatus continuously measures the resistance index (\( Ri \)), which is the pressure at the time of peak \( dP/dt \) divided by the peak \( dP/dt \) of the pressure curve in the artery.

In this study the arterial resistometr\(i\) was used to monitor the changes in \( Ri \) induced by coronary artery occlusion during percutaneous transluminal coronary angioplasty (PTCA).

Recent studies by Chierchia et al.\(^3\)\(^4\) showed that transient episodes of symptomatic and asymptomatic ischemia are associated with reductions in peak left ventricular contraction and relaxation \( dP/dt \) in more than 80% of the cases. The left ventricular hemodynamic changes that are associated with ischemia produce concomitant changes in the arterial pressure wave.\(^5\) We therefore also used the arterial resistometer to monitor and evaluate changes in arterial peak \( dP/dt \) during PTCA-induced myocardial ischemia.

Methods

Eleven consecutive patients undergoing 15 PTCA procedures were studied (table 1).

PTCA was performed with a Gruntzig coronary dilatation steerable catheter (Schneider, Zurich) introduced through the femoral artery by the Seldinger technique.\(^6\) The balloon was positioned in the coronary artery under fluoroscopic guidance by a special guiding catheter directing the balloon into the affected coronary orifice. Continuous pressure was monitored at the catheter tip and proximally in the aorta. The balloon was inflated for 30 to 60 sec under a pressure of 5 to 10 atm and was then deflated and withdrawn. No modifications in the PTCA technique were made for this study, and the entire procedure was done under conditions of coronary occlusion that are typical for clinical angioplasty.

Intra-arterial pressures were recorded via a 20 cm long catheter (Bard-I-Cath, C.R. Bard International Ltd., Sunderland, England) that was introduced via a 17-gauge needle into the femoral artery. Pressure readings were obtained by a Mennen-Medical 741 patient monitor electrocardiograph and pressure unit with a physiologic pressure transducer (Mennen-Medical...
922-122-010). The natural frequency of the transducer was 10 kHz. dP/dt was obtained by electronic differentiation that provided a linear frequency response to 100 Hz. The pressure curve obtained from the patient monitor was used as an electronic input for the arterial resistometer.

The arterial resistometer continuously monitored the Ri, which is the ratio between the pressure at the point of peak pressure dP/dt and the value of peak dP/dt.

**Mathematical model and features of the arterial resistometer.** Otto Frank’s basic and simplified mathematical analysis of the arterial pulse was used. In this model the aorta is represented by an elastic volume container, the “windkessel,” which is connected to a tube with a definite resistance to flow.

During the diastolic phase a linear relationship exists between pressure (P) and volume (V) in the “windkessel”:

\[ \frac{dP}{dt} = E \] (1)

where E is a constant.

If we assume that the pressure at the distal end of the tube (venous side) is zero, then Poiseuille’s law may be written:

\[ \frac{dV}{dt} = -\frac{P}{SVR} \] (2)

where SVR is the resistance (analogue of R on Ohm’s law).

During the systolic phase, the rate of volume change in the “windkessel” is affected by two factors: (1) the rate of inflow (from the heart), which is indicated by i and may be some function of time i (t), and (2) the rate of outflow (through the peripheral resistance), which is given (by assumption) by Poiseuille’s law, i.e., \( \frac{dV}{dt} = \frac{P}{SVR} \).

It is clear that the rate of change of volume per unit time in the “windkessel” is given by:

\[ \frac{dV}{dt} = i(t) - \frac{P}{SVR} \] (3)

Combining III with the assumption expressed by equation 1:

\[ \frac{dP}{dt} = E \left( i(t) - \frac{P}{SVR} \right) \] (4)

Because we do not know the nature of i(t), we must make an assumption. Otto Frank’s simple assumption was that the rate of outflow into the aorta is approximated by a sine function, and the relationship between SVR and the arterial pressure is expressed in the equation:

\[ \frac{dP}{dt} + \frac{E \times P}{SVR} = EA \sin Bt \] (5)

where E is a resistance factor (which is relatively constant in an individual patient and depends on the compliance of the arterial system).

At the beginning of systole, when \( \sin Bt = \sin 0 = 0 \),

\[ \frac{dP}{dt} = \max \frac{dP}{dt} \] (6)

and the solution to equation 5 is:

\[ \max \frac{dP}{dt} + \frac{E \times P'}{SVR} = 0 \] (7)

The equation by which the arterial resistometer calculates SVR is:

\[ SVR = E \times \frac{P'}{\frac{dP}{dt}} = E \times Ri \] (8)

where E is a resistance factor, \( P' \) is pressure at peak \( \frac{dP}{dt} \), and \( \frac{dP}{dt} \) is peak \( \frac{dP}{dt} \).

Linear regression analysis of several paired SVR values (thermodilution) and Ri values (arterial resistometer) showed that the intercepts of the regression lines had values below zero and the equation was changed to \( SVR = \frac{E \times Ri}{a} \), where a is the intercept of the individual regression line and E is the slope of the regression line (figure 1).

SVR calculated by the arterial resistometer showed a good correlation to thermodilution-calculated SVR by linear regression analysis \( r = .98 \). The resistometer was found to be extremely sensitive and reliable in monitoring the changes in SVR.

Calibration by thermodilution injections was not done in this

**TABLE 1**

<table>
<thead>
<tr>
<th>Characteristics of patients and PTCA procedures performed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient No.</td>
</tr>
<tr>
<td>--------------</td>
</tr>
<tr>
<td>1 2 3 4 5 6 7 8 9 10 11</td>
</tr>
<tr>
<td>Age (yr)</td>
</tr>
<tr>
<td>Sex</td>
</tr>
<tr>
<td>Previous MI</td>
</tr>
<tr>
<td>EF (%)</td>
</tr>
<tr>
<td>Type of angina</td>
</tr>
<tr>
<td>No. of vessels involved</td>
</tr>
<tr>
<td>Vessels dilated</td>
</tr>
<tr>
<td>No. of balloon dilations</td>
</tr>
<tr>
<td>ST-T changes</td>
</tr>
</tbody>
</table>

EF = ejection fraction; MI = myocardial infarction; U = unstable; S = stable; LAD = left anterior descending artery; LCx = left circumflex artery; RCA = right coronary artery.

*Second dilatation with a larger balloon.

*Second successful dilatation due to complete obstruction of LAD at first attempted dilatation.
study because we were interested mainly in changes in SVR and not in the absolute values. Therefore the results are expressed in resistance index units and not in the conventional way (dyne-sec-cm⁻²). A word of caution should be added: Ri values are not absolute values. Since the calibration constants may vary in patients, Ri was predetermined in each patient as a baseline before intervention.

Pre- and post-PTCA differences were checked for statistical significance by the paired t test.

Results

All 49 balloon inflations were associated with increases in Ri (from 38.4 ± 12 to 81.2 ± 36 × 10⁻³ sec; p < .01) and decreases in femoral arterial peak dP/dt (from 2076 ± 257 to 1327 ± 326 mm Hg/sec; p < .001) (figure 2). Typical changes in the form of the femoral arterial waveform associated with coronary occlusion are demonstrated in figure 3.

The increase in Ri correlates with the duration of balloon inflation. The time course of changes in Ri associated with balloon inflation is demonstrated in figure 4. In 42 of the balloon inflations, increases in Ri were associated with electrocardiographic ST-T changes, and in 23 they were also associated with anginal pain. No cases of angina or ST-T changes without an increase in Ri occurred. When angina was present, a further increase in Ri (to 97.5 ± 47.3 × 10⁻³ sec; p < .001) and a decrease in dP/dt (to 1218 ± 338 mm Hg/sec; p < .001) was observed (figure 5).

Two of the patients who developed anginal pain during coronary occlusion demonstrated a significant increase in Ri despite the absence of changes on standard electrocardiographic leads (table 1).

Discussion

In a recent study,² we compared SVR values determined by the resistometer with values calculated from thermodilution cardiac output measurements. The study was performed on 22 patients, and 255 SVR measurements were obtained. SVR ranged between 450 and 4400 dyne-sec-cm⁻². The patients who were studied immediately after coronary bypass operations were exposed to situations such as temperature changes, volume load, blood loss, and the effect of various catecholamines, nitroglycerin, and nitroprusside. Heart rates ranged between 48 and 160 beats/min while these measurements were obtained. The calibration constants E and a were predetermined for each patient by taking simultaneous thermodilution-calculated SVR measurements and Ri readings before (low SVR) and immediately after (high SVR) aortocoronary bypass.

Correlation of SVR measurements between the two methods was r = .98, and the correlation coefficients of individual patients ranged from .9 to .986.

In the present study no effort was made to calibrate the system against any standard method (like thermodilution), since our main concern was the change in Ri for an individual patient during the PTCA balloon inflation and not the absolute values of SVR. In this form (without calibration) the system is simpler, and Ri can be measured very easily by applying the resistometer to a reliable pressure monitoring system.

Ordinarily electrocardiography is considered the most reliable objective means of detecting myocardial ischemia.⁸,⁹ As shown here, however, the hemodynamic changes, namely the increase in Ri and the reduction in arterial dP/dt, are even more reliable. In 49 instances of arbitrary and controlled occlusion of a major coronary artery there was a hemodynamic response in all of them (table 1). In only 42 of the occlusions were electrocardiographic changes also present. The electrocardiographic changes, whenever present, were preceded by the hemodynamic changes.

Review of the literature reveals that some of the present data have already been recorded in the past.³-⁵, 10-12 Gorlin¹³ and Cannon et al.¹⁴ reported on the link between increased SVR and anginal episodes. Moreover, Gorlin¹³ postulated that vasoconstriction increases myocardial work and oxygen demand, and he
assumed that such a pathophysiologic mechanism is responsible for the development of resting angina. In the present group of patients, however, myocardial ischemia preceded the rise in SVR. This does not negate the possibility that in certain cases Gorlin’s assumption is correct.

Chierchia et al.4,10 first reported on the reduction in peak contraction and peak relaxation left ventricular dP/dt in ischemic attacks. It has also been shown by George et al.15 that the rate of pressure increase in the aorta immediately after the opening of the aortic valve bears a constant and direct relationship to the rate of pressure rise in the left ventricle during the immediately preceding isovolumetric systolic contraction. Our study, however, is the first to show that in man, arbitrary occlusion of a major coronary artery was in each case associated with a decrease of femoral arterial peak positive dP/dt. It is possible (but not proved), that dP/dt observed in this study might reflect a decreased left ventricular dP/dt.


FIGURE 3. Electrocardiographic and arterial waveform changes before (A) and during PTCA (B).
In conclusion, a new method for real-time continuous on-line monitoring of the peripheral resistance has been developed. We have shown that there is an instant rise in peripheral resistance when a major coronary artery is totally blocked. It is not yet clear whether such peripheral changes accompany all spontaneous episodes of angina. This is currently being investigated.

References
Erratum


The authors have discovered an error in the above article. Two hundred seventy-seven of the 1113 subjects reported did not meet criteria for normal stated in the Methods. The error occurred when maps were extracted from a larger computerized data base of over 4000 body surface maps.

All map analyses reported in the study have been repeated excluding the 277 subjects who did not meet criteria for normal. The age, sex, and body habitus of these 836 normal subjects are listed in table 1.

Qualitatively there were no differences between the features of maps published in the original report and the maps from the 836 normals. However, there were quantitative differences in the values of R and S amplitudes listed in table 2 of the original report. The new values are listed in table 2. Values marked with an asterisk are different from the original table at the p < .05 level. For R wave amplitudes, the differences between the original and new values reached statistical significance only for women in the 20 to 29 year age group. Statistically significant differences in S wave amplitude occurred in men in the age groups 20 to 29 years, 30 to 39 years, and 50 to 59 years and in women in the age group 20 to 29 years.

There are two reasons for only slight differences between maps of the original study and maps from the 836 normals. First, 165 of the 277 subjects incorrectly included in the original report did not meet strict criteria for normal but they had no evidence of cardiac disease, and second, the remaining 112 subjects (10% of total) incorrectly included were divided among 15 different cardiac diagnoses and therefore had small effects on the statistical study.

The legend to figure 9 was incorrect and should have read, "Locations of the peak minimum (circle) and maximum (star) QRS potentials." Copies of the revised study illustrations are available on request to the authors.

### TABLE 1

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Male subjects</th>
<th>Female subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>10–19</td>
<td>24</td>
<td>16</td>
</tr>
<tr>
<td>20–29</td>
<td>53</td>
<td>82</td>
</tr>
<tr>
<td>30–39</td>
<td>17</td>
<td>54</td>
</tr>
<tr>
<td>40–49</td>
<td>2</td>
<td>21</td>
</tr>
<tr>
<td>50–59</td>
<td>3</td>
<td>16</td>
</tr>
<tr>
<td>60–69</td>
<td>2</td>
<td>10</td>
</tr>
<tr>
<td>70–79</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>80–89</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

### TABLE 2

<table>
<thead>
<tr>
<th>Age group</th>
<th>Sex</th>
<th>Peak R wave (mV)</th>
<th>Peak S wave (mV)</th>
</tr>
</thead>
<tbody>
<tr>
<td>10–19</td>
<td>M</td>
<td>2.04 ± 0.54</td>
<td>−2.51 ± 0.67</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>1.47 ± 0.37</td>
<td>−1.79 ± 0.5</td>
</tr>
<tr>
<td>20–29</td>
<td>M</td>
<td>1.97 ± 0.56</td>
<td>−2.17 ± 0.79*</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>1.42 ± 0.40*</td>
<td>−1.74 ± 0.68*</td>
</tr>
<tr>
<td>30–39</td>
<td>M</td>
<td>1.77 ± 0.61</td>
<td>−1.84 ± 1.14*</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>1.42 ± 0.62</td>
<td>−1.52 ± 0.80</td>
</tr>
<tr>
<td>40–49</td>
<td>M</td>
<td>1.75 ± 0.46</td>
<td>−1.76 ± 1.05</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>1.42 ± 0.35</td>
<td>−1.48 ± 0.8</td>
</tr>
<tr>
<td>50–59</td>
<td>M</td>
<td>1.76 ± 0.54</td>
<td>−1.75 ± 0.77*</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>1.42 ± 0.40</td>
<td>−1.31 ± 0.88</td>
</tr>
<tr>
<td>60–69</td>
<td>M</td>
<td>1.68 ± 0.49</td>
<td>−1.16 ± 0.44</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>1.65 ± 0.64</td>
<td>−1.27 ± 0.71</td>
</tr>
</tbody>
</table>

*Different from original at the level p < .05.
Changes in systemic vascular resistance detected by the arterial resistometer: preliminary report of a new method tested during percutaneous transluminal coronary angioplasty.

R Mohr, S Rath, O Meir, A Smolinsky, Y Har-Zahav, H N Neufeld and D A Goor

_Circulation_. 1986;74:780-785
doi: 10.1161/01.CIR.74.4.780

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1986 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/74/4/780

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation_ is online at:
http://circ.ahajournals.org//subscriptions/